# APPENDICITIS*.

## Some Points in Its Diagnoses and Treatment From the View-point That Its Cause is a Strangulation Produced by Distention Behind a Ball-Valve.

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I WISH to acknowledge my indebtedness to Drs. Black and Leonard of the Hendryx Laboratory, by whose courtesy that institution was used for most of the work. The specimens of inflamed appendices of dogs are the result of some experiments undertaken to demonstrate that strangulation from over-distention of the appendix will produce appendicitis. The experiments are given in detail in the *Journal of the American Medical Association*, March 26, 1904. They were based upon the supposition that appendicitis is due to an obstruction at some point in the lumen of the appendix (this is caused by a fecal plug or a concretion lodging behind a kink or constriction), that the cavity behind this point becomes over-distended by secretion and effusion, that infection follows because this distention by pressure limits or arrests the blood supply to the mucous membrane (weakening or destroying its resistive power), and opens natural and traumatic avenues for the entrances of germs. If the obstruction gives way before necrosis has begun, a simple inflammation of the appendix follows. This may be a slight catarrh of the mucosa, causing scarcely a symptom, or it may be followed by an infiltration of all the coats of the appendix. Sometimes it will obviously be simply appendicular colic, without any inflammation following.

If the distention remains until necrosis becomes established, a more serious inflammation follows, usually with perforation, and if it remains permanently, necrosis or gangrene with perforation must follow (unless by a mere chance the contents become "encysted").

The technic in brief was as follows: Under ether, laparatomy was performed, a ligature passed about the proximal end of the appendix, subperitoneally, so as not to affect the circulation, and the appendix distended with tap water through a needle connected with a manometer to register the amount of pressure. This was varied from 75 to 150 mm. in different cases, aiming at the normal blood pressure of the dog. Usually the ligature was cut after two and a half to three hours, releasing the pressure, and the dogs were allowed to live 24 to 48 hours. At the end of this time they were killed and the specimens removed. Ten experiments were carried out on eight dogs. Most of the specimens are here; and most of them were preserved by the Kaiserling method, which preserves something of the gross appearance present in the fresh specimen, though one sees now but little of that fiery-red appearance so typical of acute inflammation of a peritoneal surface which all of these specimens presented when removed.

Nos. 2 and 3 had injected into the lumen of the appendix, in addition to the water, a bouillon culture of staphylococcus albus and coli communis. These exhibit slightly more virulent inflammation than most of the others. The others received only the water as it was drawn from the faucet in the laboratory. In Nos. 1, 7 and 8 the attempt was made to permanently occlude the appendix in the hope that necrosis and perforation would follow. In this we failed, because in a very short time, less than two hours, the ligature would cut into the tissues sufficiently to allow a leak and consequent release of pressure. We succeeded in producing so-called catarrhal appendicitis only, because all our efforts to maintain a permanent distention proved futile.

Specimen No. 4 was first simply ligated and not distended. After 48 hours there was no inflammation, and it was then again ligated and kept distended for three hours. Thirty-six hours later a typical appendicitis was present.

Specimen No. 7 was first subjected to an injection of bouillon culture of coli communis and staphylococcus albus, and neither ligated nor distended. After 48 hours there was no evidence of inflammation. It was then ligated and distended, and the ligature allowed to remain. After 48 hours more, severe appendicitis, with adhesions, developed, shown imperfectly in the specimen.

If temporary distention of a dog's appendix produces an inflammation practically identical to that frequently observed in man; if a kink or constriction is always present in human appendicitis; if a permanent occlusion of the human appendix is often found, and if extreme distention is not infrequently noted in early operations, is it not reasonable to assume that in those cases of appendicitis in which the obstruction is not present at the time of the operation, the plug or concretion has escaped into the cecum, after *temporarily* causing distention, pressure upon the blood vessels and infection?

The practical bearing of this theory upon the diagnosis and treatment of appendicitis is quite important. Some of its most valuable hints come from a careful study of the history of pain and tenderness during the first hours of the attack. If the distention comes from a sudden increase of intra-colonic pressure produced by athletic exertion, or accident, or gastro-intestinal disturbance (forcing fluid into the appendix, which, by its recoil, forces the concretion into the constriction, closing the ball-valve), the onset will be characterized by severe pain coming on suddenly. If the occluding mass is small or pliable, and is forced out through the constriction by the increasing distention, there will be a sudden cessation of acute pain. If the occlusion is permanent (that is, if the plug is too large to pass, too hard to be compressed, and fits too accurately to allow of a leak), distention will increase until the intra-appendicular pressure equals the arterial pressure (perhaps gas formation may make it exceed that), circulation will be arrested, and necrosis or gangrene will follow. After a time the nerve endings involved in that process will fail to report pain, and the patient feels relieved; but we know it is only the lull before the storm, which breaks out with redoubled fury when the gangrenous tissues give way and allow the pent-up infection to enter the peritoneum.

I believe that in many cases we can diagnose these different conditions accurately, by carefully studying the history. If I get a clear history of acute, colicky pain continuing less than an hour, or even two hours, with a fairly sudden cessation, I feel confident that the obstruction has given way, and that only a so-called catarrhal appendicitis is to be expected. There will still be pain upon moving about or from rolling of the intestines, and there will be tenderness upon pressure, but no more acute, spontaneous pain. This means inflammation in the appendix, but relief of the distention and strangulation. Again, when the pain has been severe for several hours, there has been frequent vomiting and perhaps two or three attempts at stool, there can be no practical doubt that the distention will not be relieved through anatomical exit into the colon, but by rupture of the appendix, following necrosis or gangrene. If this acute pain gradually diminishes after six to twelve hours, it is quite certain that considerable gangrene is present, usually complete gangrene of the appendix beyond the constriction. The sudden reappearance of severe pain, 24 to 48 hours after beginning of the attack, means peritonitis following. There will, of course, be many cases on the border lines in which the actual condition cannot be known; but during the last three years I have rarely failed

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to diagnose so-called catarrhal cases from perforative ones, the diagnosis being confirmed by the findings at operation or the further progress of the case.

A patient of mine recently gave a typical history of recurrent attacks of mild appendicitis in one sentence. She said: "I would have severe pain for a short time and then be 'so sore' for several days afterward." This is the characteristic history. Severe pain while the distention and strangulation continues; relief from acute pain as the obstruction escapes, but continued tenderness from the inflammation meanwhile established by the germs driven into the exsanguinated tissues. An infection once established may of course follow any one of the innumerable processes observed in appendicitis.

The treatment as suggested by this theory may, in the first stage, be likened to that of strangulated hernia. The pressure upon the blood vessels is practically a strangulation. If the strangulation is not promptly relieved, necrosis or gangrene will follow. We say, "Don't allow the sun to set upon a strangulated hernia." We may say with equal propriety, "Don't allow the sun to set upon a strangulated appendix." Could we always know that strangulation was or was not relieved, the indication would always be clear. Unfortunately, we cannot always be certain. Still, study, from this standpoint of distention as a cause, throws a flood of light upon the subject, and failure to make this diagnosis should be rare. Unfortunately again, patients so commonly fail to call the surgeon during this early period, when it is simply a question of strangulation. In a large proportion of cases it is the resulting infection (so often already a peritonitis) which claims the attention of the surgeon. If the pain has been slight and of short duration, and only slight tenderness remains, it is safe to say that the patient needs no treatment for that attack. Of course, he will probably have further attacks, and needs his appendix removed on that ground. The appendicular colic is evidence of a constriction. A plug of feces or a fecal concretion will reform and sooner or later produce another attack. But for the present he is safe with a simple catarrhal appendicitis. The possibility of dislodging this plug either into or out of the appendix by some form of taxis or massage occurs to me, but I have had no opportunity of testing it. As a matter of fact, we are rarely called early enough to apply it (within three or four hours), and many anatomical difficulties present themselves.

Operation, if performed at all, must be done before perforation or peritonitis supervene. The question of "early operation" should be settled early, within 12 hours if possible, almost certainly within 36 hours, and at least within 48 hours of the onset of symptoms. It is impossible to make rules by hours or by the severity of the pain; distention advances at such varying rates, and the virulence of germs varies so greatly. The weight given to the evidence derived from a careful study of time and pain must depend upon the judgment and experience of the surgeon. It is comparative evidence, and cannot be tested by a fixed measure. It should be only the most positive evidence that allows the case to go on with the assurance that only a mild attack without consequence is impending. When in doubt during these early hours, I advise operation every time. If the error is made of removing a catarrhal appendix early, no harm is done. It is even then a good riddance to the patient.

But appendicitis is treacherous, and more than time and pain must be considered. Sometimes in the most serious cases the patients have little or no pain during the first few hours. All the symptoms must be studied, of course, but always bearing in mind the probable stage of the strangulation. But, unfortunately again, we are so often called upon to consider this question of operation when we know the case is one of peritonitis rather than appendicitis only. It may be limited to a small portion of peritoneum, may be a walled-off abscess, but is, nevertheless, peritonitis. The removal of all the infected tissue is now out of the question, and to my mind Ochsner has conclusively proven that a much larger percentage of such patients get well if let alone than if meddled with by operation. A certain number will die with any kind of treatment; but my personal experience with both forms of procedure, though more limited than that of many surgeons here, is overwhelmingly on Ochsner's side of this exceedingly serious controversy.

One of my earliest experiences (long before I heard of Ochsner) came near converting me to the "let-alone" plan of treatment during this stage. It was a consultation 20 miles out in the country. Farmer's boy, 19 years old. Sick four to five days. He was very comfortable; evidently a localized abscess. His general condition was excellent. No suffering. But little fever or sepsis. We operated, of course, secundum artem. I don't know how I could improve the technic to-day. Severe pain followed. Next day delirium, and the following day death claimed the flower of that family. No explanation of mine could convince the family that the operation had nothing to do with the sudden change and resulting catastrophy. I could not make myself believe it. I could not see why there should suddenly be so many more deaths from appendicitis, many more than anyone ever heard of before from all forms of peritonitis put together. Dr. Thorn graphically illustrated the situation at the meeting of this society a year ago. He said: "I have practiced medicine thirty-five years. During the first twenty-two years I had only two cases of appendicitis. In the remaining thirteen years I have had two hundred cases and many deaths." Does anyone believe there were materially more cases after 1890 than before? The doctor simply diagnosed them something else, and there were not enough deaths from it to even remind him of them now.

In the presence of acute peritonitis a focus of acute infection is certain to remain after operation, and the danger of its spreading is immensely increased by the intestinal paralysis, the interference with the normal secretions of the peritoneum and the removal of the barriers already thrown up by nature's protective forces. For percentages of results by the two methods, I will only refer you to the reports of cases by Deaver and Ochsner at the meetings of the A. M. A. in 1901 and 1902. Deaver's mortality was about 15% and Ochsner's 4%. I have been wondering what effect these discussions had upon Deaver.

My experience with appendicitis is limited, compared to that of the surgeon specialist, but, from the standpoint of the general practitioner, may be of interest. I report on only those cases occurring in my practice during the last three years. During that time I have treated 28 patients, and have had the good fortune to see them all recover. Eleven cases were seen before the end of 24 hours from the beginning of the attack. Six cases between 24 and 28 hours from the onset of symptoms. In 13 cases there was peritonitis, and in seven of these striking evidence in the symptoms of a sudden perforation of the appendix. Four of these seven have had operations since, and the diagnosis of perforation was demonstrated in each case. Three of the cases developed typical general peritonitis, requiring weeks to make a complete recovery. Two of these patients (both children) were so ill that recovery seemed impossible for days. All recovered. A distinct tumor was noted in eight cases. Immediate operation in the beginning of the acute attack was advised in 11 cases, all of them before the end of 48 hours. Thirteen patients submitted to operation, four in the beginning of the acute attack, two late in the attack (opening

the remaining abscess) and seven were so-called interim operations.

All patients not immediately operated on were treated by the Ochsner method; that is, they were kept in bed and given no food or medicine by mouth as long as any decided tenderness was present. In eight cases rectal feeding was carried out for several days. Stomach lavage was employed in only one case. Ice was applied to the abdomen in one case and hot-water bag in several. Opiates, usually morphia, were given cautiously but sufficient to secure reasonable relief from pain. Cathartics were not given until gas was passing freely and the bowels had been moved by enema. In one case of general peritonitis, distention was so great that an aspirating needle was passed through the abdominal wall three or four different times and gas and liquid feces drawn off. The appendix was removed in all these except the abscess cases. All appendices removed presented evidence of constrictions. In only two were true concretions found. Both of these were distended and strangulated. One was wholly gangrenous in an early stage, and the other presented two necrotic spots, one of which broke down within an hour of its removal. The only complications encountered were a suture abscess late in the second week in a patient suffering from pulmonary tuberculosis, and a fecal fistula in one of the late operations for abscess. Besides these 28 cases, I have during these three years seen 16 patients in consultation. Most of these were treated conservatively, though many of them were given food and medicine by mouth during the acute "peritonitis" stage. Only one was operated upon during the early acute stage, three during the interim and three late in the attack, opening the remaining abscess.

There were two deaths. The first of these was a young woman about 34, suffering from pulmonary tuberculosis in an advanced stage. I saw her only once in consultation, about six days after the beginning of the attack. She then had a general peritonitis, of which she died five or six days later. She was not treated wholly by the Ochsner plan, as she was given considerable food and medicine by the mouth. The second case was seen in consultation just about 48 hours after the onset of the attack. There was evidence that perforation had just taken place, but I concurred in the opinion that probably it was so recent that immediate operation would still remove all infection. However, the patient was moved about seven miles to the County Hospital, and then subjected to operation. There was considerable free pus in the abdomen. The patient did badly under the ether, and died in 24 hours from general infection. This patient should not have been operated upon, and in a similar case I should object in future. This case is one of the first in this series, and I had not yet become so fully convinced that some of the infection is bound to remain and cause mischief in these cases. In all of these cases the idea of obstruction and distention as the cause of appendicitis has been acted upon, and has been a valuable aid. The Ochsner plan of treatment has been generally advised, and in my own cases fairly carried out; in many of the consultation cases, only partially at best.

The cases are presented as a plea for the Ochsner plan of treatment. All the patients so treated recovered. The entire series, 44 cases with two deaths, a mortality of a little over 4%, certainly emphasizes the advantage of avoiding operation during the acute stage, and altogether makes a strong argument for the conservative treatment of appendicitis, while acute peritonitis is present.

Conclusions.—1. Obstruction and consequent distention and strangulation is the cause of appendicitis.

2. The exact diagnosis can usually be made from a careful study of the history.

3. The ideal treatment is removal of the appendix before the infection extends to the peritoneum.

4. After this time no operation should be done during the acute stage.

5. Fasting, avoidance of cathartics and absolute rest insures a very low percentage of mortality in appendicitis and peritonitis.

[For discussion see the JOURNAL for October, 1904, pages 300 *et seq.*]

## MYOCARDITIS, WITH SPECIAL REFERENCE TO DISORDERED METABOLISM.

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(*Continued from page* 371, *Vol.* II.)

The next question is, What is the cause of the myocardial change? Naturally the first inclination is to ascribe it to anemia from hemorrhage, but it frequently occurs where there was neither hemorrhage nor profuse menstruation, as in the case just quoted; and so this explanation will not suffice. Certainly it is possible that anemia may result in such cases independent of hemorrhage, but this itself would simply be an evidence of disturbed metabolism which would be liable to produce changes in other tissues as well as in the blood. It has not been in my power to obtain a sufficient number of blood counts to warrant any conclusion as to the general appearance of the blood in this class of cases, but the following case will show that the anemia is not necessarily very great. In this instance the left ventricle was dilated, and a systolic murmur could be heard in the mitral and tricuspid areas as well as over the base of the heart in the second left interspace. An examination of the blood made two hours before the tumor was removed gave the following count: Red cells, 4,700,000; leukocytes, 8,000; blood platelets very numerous; hemoglobin, 66%; color index, 0.70. General appearance of cells normal. No parasites found. Differential count, eosinophiles 0.0%, large mononuclears 1.5%, lymphocytes 17.5%, neutrophiles 81%. This patient had not suffered from hemorrhage, nor did the tumor give her any inconvenience, although of considerable size; but she had it removed simply because she felt worried by the knowledge that a growth was present. The only sign of heart disturbance of which she complained was that she became dyspneic more readily upon exertion.

Examination of the heart four weeks after the operation showed that the murmurs had disappeared from the base and tricuspid area, that the mitral sound was impure, and all the cardiac sounds of less intensity than the average.

Kessler in commenting upon his case, of which an abstract has been presented, offers a theory that places the cardiac changes upon a somewhat physical basis. He believes that it is not so much the size of the tumor as its consistency that is responsible for the trouble, and just as the heart, especially the left ventricle hypertrophies during pregnancy, so do similar changes take place in patients suffering from fibroid tumors of the uterus; but they will be better marked in the latter condition, because during pregnancy the increased work of the heart is simply the result of physiological increase in the vascular area, comparatively temporary in character, as it ceases with partruition; but since the tumor consists of dense nodules with a tense capsule, it presents a marked resistence to the circulation, which is also persistent, and consequently the strain upon the heart is both greater and more prolonged. Experience makes one naturally suspicious of a theory that puts a morbid process upon such a mechanical basis. Although it would account for the improvement in the heart, which sometimes follows removal of the tumor, and even if a parallel has been drawn between this and the transitory hypertrophy of pregnancy we must